PEPTIC ULCER OF THE ŒSOPHAGUS.

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By simple or peptic ulcer of the œsophagus is understood an ulcer situated in the lower part of the œsophagus and bearing a close resemblance to the simple ulcer of the stomach. It is a condition which has received little attention in this country, although not excessively rare, and it has, therefore, seemed worth while to report the following three cases, and to give a general discussion of the disease.

For the better comprehension of the subject it will be advisable, first, to consider briefly ulcerations due to other causes, which must be ruled out before the peptic nature of the lesion can be admitted. Of these, twelve forms may be distinguished: (1) carcinomatous, (2) due to the action of corrosive substances, (3) due to foreign bodies, (4) occurring in the course of acute infectious diseases, (5) decubitus, (6) due to aneurysm, (7) catarrhal, (8) due to traction diverticula, (9) tuberculous, (10) syphilitic, (11) varicose, (12) due to thrush. The first three forms are sufficiently well known to be dismissed without further consideration.

Ulcers of the œsophagus have been reported as postmortem findings in the following acute infectious diseases: diphtheria, scarlatina, variola, typhoid fever, and pneumonia. They are due to acute inflammation, and bacteria are found present.

In diphtheria, Councilman, Mallory, and Pearce[1] found membrane in the œsophagus in twelve out of 220 autopsies, or 5 per cent. The lesions consisted of longitudinal erosions with sharply cut edges, and covered with adherent fibrinous membrane. Leube[2] states that stenosis may follow.

In scarlatina the process is a diffuse inflammation with ulceration, invading the œsophagus by extension from the pharynx; it may be followed by stenosis. The ulcers in variola arise from the breaking down of œsophageal pocks. Louis and others have called attention to ulcers in the œsophagus, occurring in typhoid fever, usually in the upper part, and accompanied by ulceration of the pharynx; the specific bacilli have not yet been demonstrated in them. They, too, may be followed by stenosis. Of lesions occurring in croupous pneumonia I have found but two instances: (1) a case of Kraus,[3] in an elderly female; numerous hemorrhagic infiltrations in the lower half of œsophagus, hemorrhagic erosions, and small ulcers near the pylorus; (2) a hitherto unpublished case from the Boston City Hospital: a man, aged fifty-four years, croupous pneumonia of the right upper and middle lobes; at the middle of the œsophagus on the posterior wall an irregular superficial ulcer, 2.5 x 6 cm., covered with diphtheritic membrane; microscopic examination showed a fibrinous membrane, with no Klebs-Loeffler bacilli. Cultures from the heart, lung, liver, and kidneys all showed the presence of pneumococci.

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[1] A Study of the Bacteriology and Pathology of 220 Fatal Cases of Diphtheria, Boston, 1901.

[2] Specielle Diagnose der inneren Krankheiten, Band i. S. 262.

[3] Die Erkrankungen der Speiserohre, p. 193.

Decubitus of the œsophagus occurs on the anterior or posterior wall, or both, corresponding in size and situation to the posterior part of the cricoid cartilage, to the pressure of which it is due. It is seen after death from wasting diseases, especially typhoid fever and tuberculosis.

The ulceration which sometimes accompanies aneurysm of the descending aorta is probably likewise due to pressure; necrosis of the mucous membrane first takes place, followed by ulceration and perforation.[1]

Catarrhal ulcers are rare; they are small and shallow, involving only the mucous membrane, with irregular edges; they are accompanied by catarrhal œsophagitis. The so-called follicular ulcers occupy the site of the mucous glands, and are small and cup-shaped.

Ulcers due to traction diverticula may show a superficial resemblance to peptic ulcers, but their situation, usually at the bifurcation of the trachea, and the presence of shrunken adherent lymph glands, reveal their true character.

Tuberculous ulcers of the œsophagus are very rare; they are usually shallow, with necrotic, purulent bases, resembling tuberculous ulcers of the mouth and pharynx. Tubercles may be found in the walls of the ulcer, and there is always tuberculosis of other organs.

Ulceration occasionally takes place in the mucous membrane over an œsophageal varix; the wall of the vein is soon perforated, and death takes place by hemorrhage before the ulcer attains a considerable size.

Ulceration from thrush is rare and occurs almost exclusively in infants.

From this brief account of the various ulcers to be met with in the œsophagus, I now pass to the consideration of the subject of this paper, peptic ulcer.

History. Ulcer of the œsophagus, resembling peptic ulcer of the stomach, was first described by Albers in 1839. From that time on, occasional cases were reported by Flower, Reeves, and others, and the existence of such ulcers was admitted by so great an authority as Rokitansky. Zenker, writing in 1878, and Birch-Hirschfeld, claimed that the ulcer in all these cases was due to carcinoma, perforated traction diverticula, and other causes, and they denied the occurrence of peptic ulcer in the œsophagus. In the following year, however, Quincke published three cases in which the peptic nature of the ulcers seemed clearly proved, and a careful microscopic examination left no loop-hole for criticism. It is owing to him that the disease became established as a definite entity. Since then cases have been reported by a number of observers, of whom Chiari, Debove, Ortmann, and Kraus have added most to our knowledge of the subject. Excluding all cases before those of Quincke on the ground of uncertainty of diagnosis, there have been reported up to the present, forty-one cases of peptic ulcer of the œsophagus; of these, eight were without autopsy, but from the clinical picture the diagnosis seems reasonably certain. I have been able to collect three other cases, hitherto unpublished, one of which came under my personal observation; this makes a total of forty-four cases, on the analysis of which the following statements are based.

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[1] Fr. Kraus. Die Erkrankungen der Speiserohre, p. 178.

ETIOLOGY. The disease in question, though rare, is probably not so infrequent as might be supposed from the small number of cases reported. A study of the records of the Massachusetts General, Boston City, and Long Island Hospitals showed that peptic ulcers of the œsophagus were noted in six cases out of 4496 autopsies, *i. e.*, 0.13 per cent.

AGE. Ulcer of the œsophagus occurs from infancy to old age; the youngest reported patient being three days old, the oldest sixty-six years. It is met with most frequently in middle age, as the following table shows:

Age.	*Cases.*
0 to 9	5
10 to 19	2
20 to 29	5
30 to 39	5
40 to 49	11
50 to 59	8
60 to 69	4
Total	40

In three cases it has been found as the source of the bleeding in hemorrhagic disease of the newborn.

SEX. Of forty cases in which the sex was specified, twenty-eight were males and twelve females; out of twelve cases with complicating ulcers in the stomach or duodenum, there were eleven males and but one female. A similar preponderance in favor of the male sex is found in duodenal ulcer, whereas ulcer of the stomach, as is well known, occurs more frequently among women. This may be due to the greater frequency of alcoholism among men, as Boas has suggested for duodenal ulcer.

Previous Disease. In order that peptic ulcer should be formed, it is evidently necessary that the cardia should be insufficient, allowing regurgitation of the gastric juice into the œsophagus. An important part is played in the etiology, therefore, by diseases accompanied by frequent vomiting, such as peritonitis, nephritis, chronic gastritis, and especially ulcer of the stomach or duodenum. In the disease last mentioned, besides the vomiting, two factors are of importance: (1) ulcer of the cardia with organic insufficiency; (2) stenosis at the pylorus, with dilatation of the stomach, which may involve the cardiac orifice, with consequent relative insufficiency. The importance of the second factor is shown by the presence of stenosis of the pylorus or duodenum in eight of the cases with autopsy, or 23 per cent.

In nephritis, besides the frequent vomiting, the deleterious action of the uræmic poison on the tissues is probably effective. Disease of the kidneys was found in four cases; it was probably present oftener, as in many instances the condition of the kidneys is not mentioned.

That peptic ulcer may result from mercurial poisoning seems probable from the following case occurring at the Massachusetts General Hospital (service of Dr. R. H. Fitz): Minnie P., female, aged thirty-seven years, entered the hospital November 26, 1901, with general œdema and advanced failure of compensation. There was a history of frequent vomiting lately. She received, for purposes of diuresis, 63 grains of calomel in seven days, combined with opium and digitalis. The stools became frequent and bloody, and there was hemorrhage from the gums also. Death took place December 5th. At the autopsy were found a dilated heart, with double lesions of the mitral and aortic valves; chronic passive hyperæmia of the kidneys, lungs, liver, and spleen, and severe diphtheritic colitis; in the lower part of the œsophagus were four or five shallow ulcers involving the mucous membrane only, with reddish, finely granular bases, and irregular outlines, the largest 12 mm. in diameter; the surrounding mucous membrane was healthy; the stomach was normal. No microscopic examination of the ulcers was made. The lesions in the intestine corresponded with those found in acute mercurial poisoning.

In this case it must be admitted that it is possible the ulcers were due to the action of mercury, either alone or combined with passive hyperæmia. In view, however, of the situation in the lower part of the œsophagus, and of the opportunity offered for the action of the gastric juice by the frequent vomiting, it seems reasonable to assume that the mercury acted merely as a predisposing cause, and that the ulcers were peptic.

Chlorosis, which has been supposed to favor the development of ulcer in the stomach, probably plays no part in the production of œsophageal ulcer, for it was not present in any of the cases.

OTHER FACTORS. The abuse of alcohol deserves mention among the predisposing causes, occurring with considerable frequency. Alcohol probably acts in two ways: first, by irritation of the œsophagus, and second, by the production of gastritis, with vomiting and insufficiency of the cardia.

Various lesions of the heart and vessels have been found present, and may be of some importance in etiology, through disturbance of nutrition in the œsophagus. Among them arteriosclerosis, aortic aneurysm, hypoplasia of the aorta, and myocardial degeneration may be mentioned. In a case of Carstens with amyloid disease, it is uncertain whether the diseased condition of the vessels or the accompanying nephritis was effective.

The possibility of a traumatic origin must be considered next. Ortmann has reported cases in which the first symptom came on after swallowing food of an irritating nature, *e. g.*, sour wine, and in which he assumed that spasm of the œsophagus took place, producing a tear in the wall, which became a starting point for the ulcer. It is possible, however, that the ulcer already existed in a latent form, and the question cannot be considered settled.

The relation of the acidity of the gastric juice to ulcer is an interesting one, but unfortunately the point was noted in only seven instances. Of these, four were complicated with ulcer of the stomach (or duodenum); free HCl was "present" or increased in three, diminished in one case. In the three cases in which the ulcer was in the œsophagus alone, HCl was "present" in two cases, increased in one. The figures are too few for any deductions, but one would expect hyperacidity to be the rule, as in gastric ulcer.

Large abdominal tumors were present in three cases, in one of which there was also cancerous peritonitis. Quincke expressed the opinion that here, by the pushing up of the diaphragm, a kinking of the œsophagus was produced, which in turn caused circulatory disturbances favorable to ulcer formation. Acute and chronic peritonitis, each noted in two instances, might be efficacious in two ways, by causing vomiting, and by pressure on the diaphragm (meteorism).

Fischer[1] has reported ulceration of the œsophagus after extensive burns, analogous to ulcer of the duodenum under like circumstances. Probably thrombosis or embolism is the primary event here, due to disintegration of the red cells of the blood, or possibly to septic processes.

It remains to discuss the possibility of a nervous origin for the ulcer. Guitéras has reported the following case, which he considered to be peptic ulcer due to hysterical ischæmia and merycism: A woman, aged forty-four years; had nervous prostration nine years before; two years later, difficulty with swallowing, especially with liquids; globus hystericus. Eighteen months before entrance to the hospital a similar attack, but more severe, was cured temporarily by the passage of bougies. Return of symptoms, with complete inability to swallow; emaciation and death. At autopsy the œsophagus, from a point four and one-half inches below the cricoid to the cardia, was riddled with ulcers, pin-head to pea-sized, for the most part superficial; opposite the bifurcation of the trachea were two larger ulcers, which had perforated into the mediastinum; numerous pigmented scars, apparently of healed ulcers, were also present.

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[1] Quoted by Huwald, p. 24. (See bibliography.)

The earlier symptoms in this case point clearly to nervous spasm of the œsophagus, and in this condition it is known that secondary dilatation may take place, with stagnation of food in the œsophagus. In a few of these cases[1] numerous thickly set ulcers have been found, very similar to those described by Guitéras. In view of these facts, the peptic character of the ulcers in his case seems very doubtful, and I have, therefore, not included it in this series.

There are no other cases in the literature which point to a nervous origin of œsophageal ulcer, and from what we know of gastric ulcer such an origin does not seem probable.

PATHOLOGY. Simple ulcer of the œsophagus resembles very closely, both in gross and microscopic appearances, simple ulcer of the stomach. It is situated, as a rule, in the lowest part, close to the cardia, as one would expect from the peptic origin. Usually it stops short at the cardia; in six cases, however, or 17 per cent. of the thirty-six cases which came to autopsy, it involved the adjacent part of the stomach also, and the appearance of the ulcer pointed to a primary seat in the cardia, with invasion of the œsophagus by direct extension. It may reach as high as the bifurcation of the trachea, but single ulcers situated at this level (as in Part's case[2]) are to be viewed with suspicion, as it is not likely that the gastric juice would exert its action at such a distance from the stomach. Of the seven cases in which definite statements are made as to the situation, the ulcer was in the anterior wall twice, in the posterior wall three times, on the left side twice, and three times on the right; the right posterolateral wall seems, therefore, to be the seat of predilection. The size varies greatly, from that of a pea or smaller to ulcers involving the entire surface from the cardia to the bifurcation. In most of the cases (81 per cent.) but a single ulcer is present; in others they are very numerous. It is probable that some of the large, irregular ulcers arise by confluence. The smaller ulcers are mostly round or oval, the larger are irregular, often girdling. If very recent, the loss of substance is slight, involving only the mucous membrane, usually irregular in outline, with clear cut edges and red, granular base. Older ulcers usually lay bare the muscular coat, and may show the typical punched-out appearance and funnel-shaped base of the gastric ulcer; if of long standing, the base is smooth and the edges may be undermined. The surrounding mucous membrane is healthy as a rule; there may be dilatation of the lower œsophagus, even when no stenosis is present.

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[1] See A. Neumann. Centralbl. f. d. Grenzgebiete d. Med. u. Chir., iii., 253; and A. Schmidt. Münch. med. Woch., 1899, p. 304.

[2] Lancet, 1857, p. 167.

The ulcer, like its namesake in the stomach, has a strong tendency to perforation, which took place in six cases: twice into the right pleural cavity, once into both, with double pneumothorax, and once each into the aorta, pericardium, and lesser omental cavity. In Janeway's case, without actual perforation, there was inflammation of the periœsophageal tissue, pericarditis, and pleurisy.

Microscopically, there are found the usual appearances of chronic inflammatory tissue, with the absence, of course, of evidence of cancer, tuberculosis, etc. In two cases, thrombosed arteries were found in the base. In Fraenkel's case, Stoerk found at the edge of the ulcer glands identical with those found in the stomach; Schaffer[1] and others have noted the presence of such glands in the normal œsophagus; there were similar findings in my first case. Kraus, however, sought them in vain in three instances.

Healing takes place frequently, with the production of a scar. Scars were found, either alone or in conjunction with fresh ulcers, in nine cases, or 25 per cent. They may be puckered and radiating, as in the stomach. If small, they do no harm; the larger ones by contraction produce stenosis. In one case (Ortmann) a carcinoma of the œsophagus was found developing from the scar of an old peptic ulcer; I have seen the specimen from a similar case, at the Pathological Institute in Vienna.

The cases of peptic ulcer may be divided into three groups: (1) those in which the œsophagus alone is involved; (2) those showing extension from the cardia; and (3) those with ulcers in other parts of the stomach or in the duodenum. The first group is much the largest, with twenty-two cases, or 61 per cent. of the cases with autopsy. The second comprises six cases (17 per cent.) and the third twelve cases (33 per cent.). The last two groups cannot be sharply separated, because in several instances both the cardia and other regions of the stomach were involved. The distribution of the ulcers in the third group was as follows: anterior wall, one case; posterior wall, one case; pylorus, five cases; duodenum, five cases. Stenosis of the pylorus and of the duodenum, with dilatation of the stomach, were each present four times. In the case of Kraus, besides stenosis of the duodenum, there was an hourglass stomach. In three instances death was due to the perforation of a gastric ulcer.

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[1] Beiträge zur Histologie menschlichen Organe, Sitzungsberichte der Wiener Akademie, October, 1897, Band. cvi., 3 Abth.

Hemorrhagic infiltration of the œsophagus, with numerous erosions in the stomach, was reported by Kraus in a case of pneumonia. From the resemblance of the lesions to those seen in the stomach in certain cases of septicæmia, an infectious origin seems probable, and I have, therefore, classified it among the ulcers of acute infectious diseases.

Ulceration of the œsophagus in hemorrhagic disease of the newborn demands a separate consideration. There are three such cases in the literature. The first (Henoch) showed a deep ring-shaped ulcer, 2 cm. long, just above the cardia, with gray-white, infiltrated base; the throat was normal (no diphtheria). In Spiegelberg's case there was a pea-sized area of hemorrhagic infiltration at the junction of the cardia and the œsophagus, with superficial necrosis; microscopic examination showed no lesion of the vessels; blood-cultures during life were sterile. In the third case (Meyer) the lesion consisted of an ulcer immediately above the cardia, with an elongated thrombus measuring 6 x 7 x 12 mm., tightly adherent. Microscopic examination showed inflammatory infiltration of the base and edges of the ulcer, with streptococci and long bacilli in these tissues, and also in the circular muscular coat; sections of the liver, spleen, and kidneys showed no bacteria; no cultures were made. The stomach and intestines were normal in all.

The presence of bacteria in the tissues in the last case, and of hemorrhagic infiltration in the second, are strongly suggestive of a bacterial origin, a view that is often taken nowadays of hemorrhagic disease of the newborn in general. The lesions just described resemble those found in the stomach and duodenum in a few other cases of this disease. The sequence would appear to be hemorrhagic infiltration (due to capillary embolism), necrosis, then digestion by the action of the gastric juice, with ulceration.

LESIONS IN OTHER ORGANS. Of these the most interesting are the abdominal tumors, which were present as the primary disease in three cases; curiously enough, they all arose from the ovary Whether this is anything more than a coincidence must be left to the future to decide. The nature of the growth was as follows: carcinoma of the ovary with cancerous peritonitis (Quincke), enormous cyst (Quincke), fibroma the size of a man's head (Sabel).

Chronic interstitial nephritis was noted in four cases, in one of these combined with amyloidosis of the liver and spleen. Tuberculous peritonitis and acute general peritonitis were each present twice; the latter was due to perforation of a gastric ulcer and of the gall-bladder respectively. Cirrhosis of the liver and phthisis were each present in two cases. In the case of Dasse, there was an aneurysm of the aorta, the size of a hen's egg, adherent to the œsophagus. The aneurysm was situated at the fourth dorsal vertebra, the ulcer 4 cm. above the cardia; the distance between the two renders any direct causal relationship doubtful.

PATHOLOGICAL DIAGNOSIS. This may be summed up as follows: an ulcer in the œsophagus is to be regarded as peptic when it presents the appearances already described, is situated in the lower part of the œsophagus, and none of the other causes of ulceration are present. The finding of similar ulcers in the stomach or duodenum strengthens the diagnosis.

PATHOGENESIS. As already mentioned, the first requisite for the formation of the peptic ulcer of the œsophagus is an insufficiency of the cardia. This may be organic in the cases in which the ulceration is at the cardia; in the rest it is functional. Normally the cardia is closed, owing to the tonic contraction of the circular fibres. Relaxation takes place through inhibitory impulses from the vagus fibres, and occurs with the act of swallowing, and with pyrosis, belching, and vomiting. Another source of insufficiency is a decrease in tone of the cardiac sphincter, similar to that which occasionally takes place at the pylorus. In this connection, it should be mentioned that a rhythmical relaxation of the cardia has been observed in some animals during digestion, with regurgitation of food into the œsophagus.

Evidently insufficiency must occur frequently without producing ulcer; for the formation of an ulcer something else must be added. It is generally admitted for gastric ulcer, that the first step is a necrosis of the mucous membrane, which then becomes digested away by the action of the gastric juice, with resulting ulcer formation. But why the necrosis takes place has never been satisfactorily explained. The prevailing theory is that it is due to circulatory disturbances of one sort or another. Infectious thrombosis has been assumed as a cause, and probably holds good in some cases of hemorrhagic erosion, but has not been shown to occur in peptic ulcer. Thrombosis of an artery in the base of the ulcer has been demonstrated in two cases (Zahn and first case of Tileston), but was probably secondary in the first, and certainly in the second, in which there was also erosion of the arterial wall. Quincke's theory of kinking of the œsophagus, owing to upward pressure on the diaphragm, would account for only a limited number of cases. That hyperacidity of the gastric juice plays a part in preventing healing of the ulcer seems probable, but the data on this point are insufficient for definite conclusions. In short, the real cause of peptic ulcer of the œsophagus, as is the case with gastric ulcer, remains hidden.

Before passing to the description of the clinical picture, I have to report three new cases, which possess several interesting features.

CASE I.—From the service of Dr. J. J. Minot, Long Island Hospital, Boston.

Catherine C., aged fifty-eight years, widow, was admitted on March 23, 1905, in a moribund condition, and died one hour afterward. No physical examination was made. The history obtained from a friend later was unsatisfactory. Fifteen years ago amputation of the left leg was performed; cause not ascertained. Eight years ago she was in a hospital with "heart trouble." Cough and dyspnœa for several days previous to admission; no symptoms referable to the œsophagus.

Autopsy: Ninety-one hours postmortem. Dr. G. B. Magrath.

Anatomical Diagnosis: Chronic adhesive pleuritis; hydropneumothorax; atelectasis; acute vegetative endocarditis (mitral); fatty infiltration of the myocardium; ulcer of the œsophagus with perforation into the pleural cavity; chronic perisplenitis; fatty metamorphosis of the liver; chronic nephritis; hemorrhagic infarction of the kidneys; hemorrhagic pachymeningitis; general arteriosclerosis (slight).

Body that of a woman, aged fifty-eight years; length 142 cm.; well-developed and nourished. On the trunk and inner aspect of the thighs a brown, macular, and somewhat diffuse pigmentation, emphasized over the abdomen and at the axillæ; the skin of the right lower leg and ankle shows faint, whitish, irregular scars.

Subcutaneous fat 3 cm. in thickness; muscles fairly well developed, rather fatty. Chest wall prominent and convex; height of diaphragm, sixth rib on right side, fifth rib on left. Puncture of the wall of the right chest is followed by the escape of numerous bubbles of air or other gas.

Pleural Cavities: Left: interrupted by rather numerous dense fibrous bands, and contains 500 c.c. of clear serous fluid; right: contains 1300 c.c. of yellowish, somewhat turbid fluid, with strings of fibrin; a few fibrous adhesions at the apex and base; the inrne wall, just above the diaphragm, presents an elliptical opening, 3 x 6 cm., apparently communicating with the œsophagus, and with edges slightly rounded; the adjoining pleural surface a little roughened.

Œsophagus: On the right lateral and posterior walls, at a level 2 cm. above the opening into the stomach, an irregularly oval ulcer, its vertical axis 3.5 cm., the transverse 2 cm.; the edges are rounded, somewhat inverted, and perfectly smooth; the floor is somewhat uneven, gray, mottled with yellow, and fairly firm; the depth is about 1 cm.; the edges are extensively undermined, and the left border a good deal indurated; in the deepest part of the ulcer is the opening into the pleural cavity, already described. The neighboring lymph nodes and those along the lesser curvature of the stomach are of the size of beans, upon section, extensively pigmented but otherwise negative.

Pericardial Cavity: Normal.

Heart: Weight, 305 grams; epicardial fat fairly abundant; on the epicardium of the right ventricle fairly numerous punctate hemorrhages; myocardium pale brown-red, and rather limp; that of the right ventricle shows some invasion of the epicardial fat. Wall of left ventricle 1.3 cm., of right 0.3 cm. in thickness. Mitral valve 10, aortic 7, pulmonary 7, tricuspid 11 cm. in circumference. Depth of left ventricle 9 cm. Cavities of both ventricles a little dilated. Both curtains of the mitral valve show nodular vegetations along the line of apposition, grayish, translucent, and granular, and about 0.4 cm. in elevation; on the anterior curtain there is also a "cauliflower" excrescence. The right posterior cusp of the aortic valve shows a calcareous nodule at its base. Pulmonary and tricuspid valves normal. Coronary arteries fairly smooth.

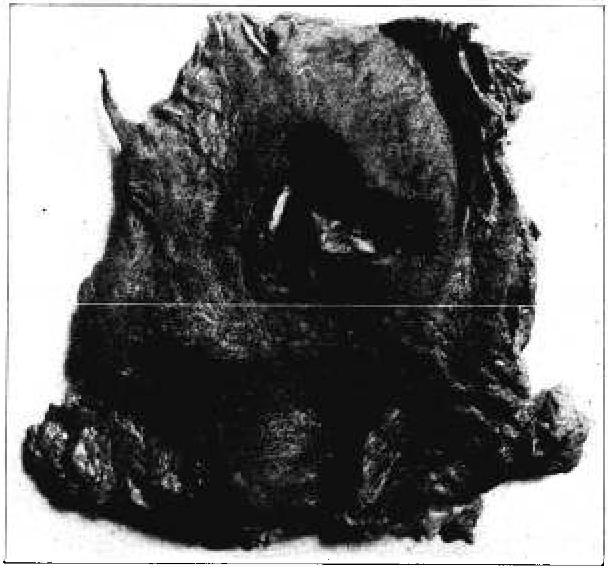
Lungs: Left: moderately crepitant throughout; slight scarring at the apex; the lobes interadherent; upon section, gray-red, under pressure yielding a little frothy fluid; bronchial lymph nodes black. Right: of diminished volume; lies back in the pleural cavity close against the inner wall; slaty gray; the pleural surface for the most part smooth, with here and there an attached fibrous tag and shred of fibrin; the upper and middle lobes interadherent, the apex without scars; the upper lobe somewhat crepitant and downy; the lower lobe flaccid and non-crepitant; careful inspection reveals no opening upon the pleural surface, or in the walls of the primary bronchi; upon section, similar to the left lung.

Abdomen: Peritoneal cavity interrupted by fibrous adhesions, uniting the spleen and the surrounding structures, and by others in the region of the foramen of Winslow. Spleen: weight 120 grams; bound down by adhesions, extensively fragmented upon removal; garnet red; markings indistinct; pulp seemingly increased. Stomach normal. Intestines: the ileum in its upper portion shows moderate injection of the veins and is of a general pale brick-red color; upon section, containing thin, semifluid material of the same color; colon empty. Vermiform appendix normal. Liver: weight 1200 grams; extensively dislocated downward; the surface in general smooth; general color pale brown; upon section, lobular markings distinct; the centres brown and a little depressed, the borders pale brown; consistence friable. Kidneys: weight of both 210 grams; capsule somewhat adherent to a surface marked by numerous shallow, in some instances, fairly deep scars, often linear, and giving to the organ a lobulated appearance; general color grayish-red; left: upon its posterior border, about midway between the poles, presents a cyst, 4 cm. in diameter; right: upon section, shows within the cortex here and there small dark-red splotches, 2 to 3 mm. across; cortex 0.5 cm. in average thickness; reddish-brown; glomeruli visible; consistence increased. Genitalia: uterus negative; appendages show senile atrophy. Aorta shows an inner surface mainly smooth, with a few slightly elevated non-calcified patches of yellowish thickening in the abdominal segment and in the arch. Gall-bladder, pancreas, adrenal glands, and bladder not remarkable.

Cranium: Sinuses normal; the dura of the right occipitoparietal region upon its inner aspect presents rather numerous, irregular, slightly elevated, dark-red areas, for the most part 1 cm. across, to some extent, confluent; the overlying inner surface smooth. Brain: vessels of the base for the most part normal; the internal carotid arteries at the turn showing a little thickening; upon section, negative; the subdural space contains a little more than a normal amount of fluid. Spinal cord not examined.

Histological Examination. Œsophagus, ulcer: section of a segment through the left side of the ulcer, including the floor to its centre: at the free edge of the ulcer there are villi devoid of epithelium; here the submucosa contains a rather large number of widely dispersed plasma and lymphoid cells; the villi of the glands at the turn of the edge are lost in a zone of necrosis, about 0.5 mm. in breadth, which extends along the entire surface of the ulcer; the loss of substance involves the muscle coats and the connective tissue beneath them; outside of the zone of necrosis the tissues contain fairly numerous lymphoid and plasma cells, with occasional leukocytes and young connective-tissue cells, and in places newly formed bloodvessels. At several points small arteries lead into the surface of the ulcer, and one of them presents erosion of its wall upon the side toward the ulcer and is partially occluded by a thrombus which is continuous with the floor of the ulcer. Beneath the surface of the deepest part of the ulcer is fat tissue which has been largely replaced by a rather loose-meshed connective tissue, containing large, sometimes stellate cells in a fibrillar stroma; here and there,

FIG. 1

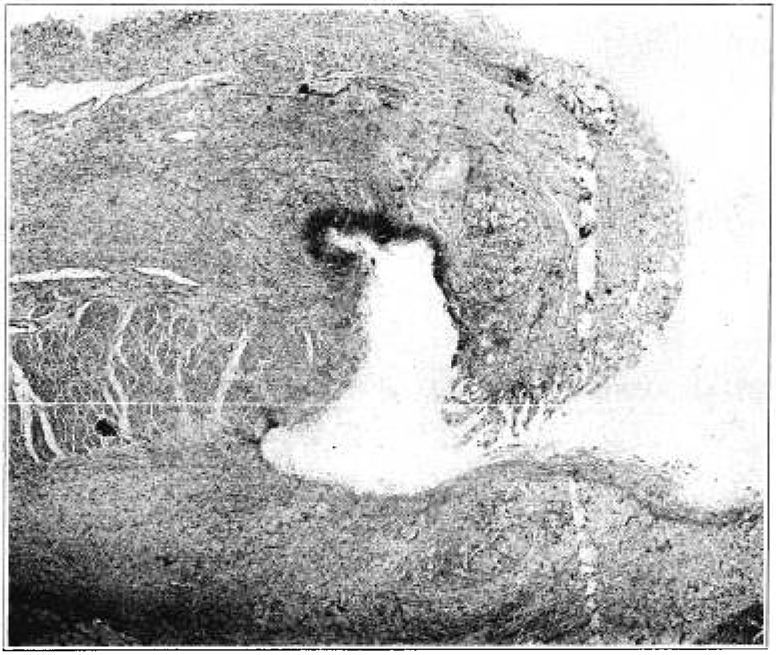


Gross appearances of the ulcer of the œsophagus (4/5 natural size), showing the perforation.

particularly around the bloodvessels, are groups of lymphoid and plasma cells. Near the edge of the ulcer, in the submucosa, is a large group of glands with columnar epithelium, resembling mucous glands in structure.

A section of the œsophagus a short distance above the level of the ulcer shows the following appearances: muscular coats normal; epithelial layer absent (postmortem change); the mucous surface is represented by villous-like stalks of connective tissue. In the submucosa are cross-sections of several ducts.

FIG. 2



Microscopic section of the ulcer of the œsophagus, showing the overhanging edge.

Kidney: There are many small, irregular areas of slight connective tissue-increase, and numerous masses of connective tissue extending inward from the capsule, like trabeculæ. In these regions there are many atrophied tubules and sclerosed glomeruli. In some there is abundant infiltration, with lymphoid and plasma cells. There is slight endarteritis and moderate perivascular increase of connective tissue. Many Henle and collecting tubules are filled with hyaline material. Histological diagnosis: chronic interstitial nephritis.

The accompanying illustrations (Figs. 1 and 2) give a good idea of the gross and microscopic appearances of the ulcer. The photomicrograph is by L. S. Brown, of the Clinico-Pathological Laboratory of the Massachusetts General Hospital.

In this case the typical appearance of the ulcer and the situation make the diagnosis of peptic ulcer certain. That the perforation took place during life is shown by the existence of acute inflammation of the pleura of the corresponding side. Interesting features are the apparent latency up to the time of perforation, the presence of chronic nephritis, and the close resemblance of the mucous membrane about the ulcer to that normally found in the stomach.

Case II.—George R., a cabman, aged fifty-nine years, was sent to the Massachusetts General Hospital (service of Dr. W. L. Richardson), on June 15, 1902, by Dr. Roscoe E. Brown, to whom I am indebted for the history. The patient had been failing for some months, suffering from retention of urine due to stricture of the urethra. His digestion was upset, and for a few weeks previous to entrance to the hospital he had difficulty in swallowing solid food; during the last two days he could take nothing but liquids. There was a good deal of pain and vomiting. The patient on entrance was semicomatose, delirious, and apparently in great pain. From time to time he vomited a thin fluid tinged with blood. Death took place in coma fifteen hours afterward.

Autopsy, twenty-two and one-half hours postmortem. Dr. J. H. Wright.

Anatomical Diagnosis: Chronic interstitial nephritis; extensive ulceration of œsophagus; chronic interstitial orchitis; focal tuberculosis of the left kidney; slight arteriosclerosis of the aorta.

The lower end of the œsophagus for a distance of 11 cm. is denuded of mucous membrane, except for a few small islands, and of a dirty brown-yellow color. The ulceration is very shallow, not involving the muscularis. The bordering mucous membrane has smooth edges, and is not discolored. At the upper margin the loss of substance is continued upward in the form of two irregular streaks; the longest is 5 cm. No microscopic examination was made. The mucous membrane of the stomach of a dirty slate color.

Kidneys: left, 36 grams; right, 97 grams. Capsule adherent; cortex very narrow; normal markings absent. In the left, several sharply defined, opaque, firm, yellowish masses, the largest 3 mm. in diameter.

In this case the ulceration is more acute in character than in the preceding, and probably extended rapidly during the last few days, when the patient was in the uræmic state. In spite of the lack of microscopic examination there can be no doubt of the diagnosis.

Case III.—Martha H., aged twenty-seven years, widow. Admitted to the Massachusetts General Hospital, April 30, 1898 (service of Dr. F. C. Shattuck). One year before, stomach trouble with anæmia. For two weeks, gastric pain, vomiting, and prostration. This morning taken suddenly with severe abdominal pain. An immediate operation (Dr. F. G. Balch) revealed general peritonitis, but no perforation could be found. Death took place the next day.

Autopsy, three and one-half hours postmortem. Dr. J. H. Wright.

Anatomical Diagnosis: Ulcus ventriculi with perforation; general acute fibrinopurulent peritonitis; slight ulceration in the œsophagus; obsolete tuberculosis of mesenteric glands; œdema of pia; laparotomy wound.

Peritoneal cavity full of yellow, cloudy fluid containing flocculi. Stomach adherent to left lobe of liver. Ulcer in anterior wall of stomach, midway between pylorus and cardia, presenting irregular perforation 1 cm. in diameter, with smooth edges. This opening leads to an abscess cavity the size of a bean, between the stomach and liver, surrounded by chronic inflammatory tissue. No evidence of cancer about the ulcer.

Œsophagus: In the œsophagus, several losses of substance; ulcers shallow, with ill-defined edges and rather dark-red, granular bases. Microscopic examination of œsophagus: in places the epithelium absent, exposing the submucosa or membrana propria, in which there is a marked infiltration with plasma cells.

In this case, therefore, we see a chronic ulcer of the stomach with perforation, resulting in localized, and later general peritonitis. The œsophageal ulcers are of very recent date, judging from their superficial character and fresh granulations. They probably occurred some time after the first perforation of the ulcer of the stomach. The infiltration with plasma cells shows that the process took place during life.

I now return to the general discussion of the disease and pass to the clinical aspect.

SYMPTOMS. In a large proportion of the cases the ulcer has not been suspected during life, being either entirely latent, or giving rise to no symptoms sufficiently characteristic for a diagnosis. In another group, it has remained latent up to the time of perforation. The symptoms vary in different stages of the disease according as the ulcer is open or healed. The most prominent manifestations of the stage of ulceration are five, namely, pain, dysphagia, vomiting, hæmatemesis, and perforation. Owing to their importance it will be best to consider each in detail. The cases in the newborn are not included in this discussion. The only symptoms they present are hæmatemesis and melæna.

Pain: This is the most constant symptom. Out of thirty-four cases in which the history is given it was present in 82 per cent. The pain was of such a character as to suggest an origin in the œsophagus in 60 per cent. of those cases in which it was accurately described. The characteristic location is at the xiphoid cartilage, or beneath the sternum, radiating to the back between the shoulder-blades. In some cases it was referred by the patient directly to the lower end of the œsophagus, in others, to the front of the chest. In a few instances the pain was epigastric, although the ulcer was situated in the œsophagus only. Tenderness to pressure has been observed in the epigastrium and over the lower sternum; no mention is made of tender points in the back analogous to those frequently found in gastric ulcer.

The relation to food is important. As a rule, the pain occurs either during the last part of the act of swallowing or immediately afterward, in contrast to the later onset of pain in gastric ulcer. It varies from a slight burning sensation to a pain so severe as to prevent the patient from eating, and to require morphine. It is usually paroxysmal, with intervals of partial or complete relief. The passage of bougies may cause great pain.

Dysphagia: Difficulty in swallowing was present in 52 per cent. of the cases. In the early stage the method of production is as follows: the food in passing over the surface of the ulcer causes pain, which in turn by reflex action causes spasm of the œsophagus, to which the dysphagia is due. At first the patient merely experiences an obstruction at a certain point, which is soon overcome, and the food enters the stomach; later, however, the spasm is so severe that regurgitation takes place. The dysphagia and obstruction to the passage of sounds may be intermittent, probably owing to variations in the amount of spasm. In a case of Quincke, of ulcer involving both cardia and œsophagus, there was a pouch formed in the wall of the latter, which when filled with food, or entered by the sound, acted as a valve, obstructing the lumen; vomiting, however, was always possible.

Vomiting: This symptom was noted in 71 per cent. of the cases. In some cases it is due to complicating lesions in the stomach, such as ulcer, chronic gastritis, pyloric stenosis; in others it is apparently reflex, and due to the pain occasioned by the passage of food. The vomiting in some cases is of rare occurrence in others, almost incessant. It takes place usually soon after meals.

Hemorrhage: This was observed during life in eighteen cases, or 53 per cent. of the cases with a clinical history. In two others the stomach was found full of blood at autopsy, death occurring before hæmatemesis could take place. In seventeen cases the blood was vomited, in one bloody stools alone are mentioned. There were both hæmatemesis and melæna in seven cases. The blood when vomited, as a rule, presents the familiar "coffee-grounds" appearance; if the hemorrhage is profuse, however, and vomiting occurs early, it may be still a bright red. The source of the hemorrhage is found to be erosion of an artery in the base of the ulcer; in one instance (Eversmann) the aorta itself was perforated. The hemorrhage is usually repeated, and frequently profuse, causing severe anæmia. In the case of Zahn the red count fell to 950,000 and a diagnosis of pernicious anæmia was made. There is no criterion by which one can tell, from the appearance of the blood, whether it comes from an ulcer in the œsophagus or in the stomach. Curschmann,[1] however, has noted that in hemorrhage from a ruptured œsophageal varix the blood usually gushes out without the nausea and straining which accompany gastric hemorrhage. It seems probable that this form of hæmatemesis, when accompanied by signs of ulcer, would point to the œsophagus as the origin rather than to the stomach.

Perforation: This ominous event was of quite frequent occurrence, being noted in six cases, or 14 per cent. of the total number. It was generally fatal. With perforation into the pleural cavity the patient is suddenly seized with intense pain in the chest, dyspnœa, and collapse, with the signs of pneumothorax or hydropneumothorax. The symptoms differ from those of the so-called spontaneous rupture of the œsophagus, in that subcutaneous emphysema is absent. Perforation below the diaphragm gave rise to circumscribed peritonitis of the lesser omental cavity.

Other symptoms mentioned are: dyspepsia, with anorexia, nausea, belching, pyrosis, inanition, constipation, and increased thirst.

SYMPTOMS OF THE SECOND STAGE. In those cases in which the ulceration has not been extensive this stage is not accompanied by symptoms; usually, however, the evidences of a benign stenosis of the œsophagus are present. There is a gradual onset of inability to swallow, involving first solid and then fluid food, and regurgitation takes place, at first immediately, and later, when dilatation has occurred, a considerable time after eating. With the stomach tube as much as a litre may be removed from the dilated œsophagus; that it does not come from the stomach is shown by its alkaline or neutral reaction, with absence of HCl and ferments; milk is returned uncurdled. On passing a sound an obstacle is encountered which may be impassable except for the smallest calibres.

COURSE OF THE DISEASE. The onset is usually gradual, beginning with pain and dysphagia; frequently dyspepsia or the symptoms of gastric ulcer precede. In other cases the ulcer remains latent up to the occurrence of hemorrhage or perforation; when ulceration takes place as a terminal event in other diseases there may be no symptoms pointing to the œsophagus. The course is often remittent, periods of comparatively good health alternating with attacks of pain, vomiting, and hæmatemesis. The duration, including the stage of stenosis, varies from a few days up to twelve years; in 63 per cent. it was under six months. Death took place most frequently from perforation or hemorrhage; other causes were inanition, perforation of other organs (stomach, gall-bladder), and chronic wasting diseases.

DIAGNOSIS. The diagnosis of peptic ulcer of the œsophagus is difficult, and in many cases impossible; in others it may be made with a considerable degree of certainty. Peptic ulcer should be suspected in all cases with symptoms pointing to ulceration in the lower part of the œsophagus. The diagnosis is practically certain, if other causes of ulceration can be excluded, and there are dysphagia, pain during or soon after deglutition, situated under the sternum or between the shoulder-blades, hæmatemesis or melæna, and obstruction in the lower part of the œsophagus. Symptoms of present or past ulcer of the stomach or duodenum are very strong confirmatory evidence. Tenderness over the lower part of the sternum, and perhaps, also, behind near the ninth dorsal vertebra, should be looked for. Up to the present the œsophageal appearances have been recorded in but a single case, that of Starck.[1] He saw in the left anterior quadrant, 38 cm. from the teeth, a whitish ulcer with infiltrated edges and dirty, dark-red base. When touched, it bled readily, and was very painful. He made the diagnosis of carcinoma, but the complete recovery showed that he had to do with peptic ulcer. It seems probable that in the future the œsophagoscopic diagnosis will be possible for the expert, supplemented, if necessary, by excision of a piece of tissue for microscopic examination. The detection of hemorrhage may be aided by the use of the guaiac test for blood in the stools. Great care should be used in the passage of sounds, owing to the danger of hemorrhage.

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[1] Deutsche med. Woch., April 17, 1902.

Differential Diagnosis. Peptic ulcer of the œsophagus is to be distinguished from ulcer of the stomach or duodenum, and from ulcers of the œsophagus due to other causes.

From ulcer of the stomach it differs in the presence of dysphagia and in the character of the pain, which in gastric ulcer occurs at a later period after eating, and is referred to the epigastrium and lowest dorsal region, but not to the chest. Tenderness over the sternum points to the œsophageal origin; epigastric tenderness is equivocal. Ulcer at the cardia gives rise to the same symptoms as ulcer in the lowest part of the œsophagus, and cannot be distinguished from it.

Passing to the consideration of ulcers of the œsophagus which are not peptic, those occurring in the course of acute infectious diseases heal promptly, and, therefore, are not likely to cause confusion. The decubitus of the œsophagus may be seen with the laryngoscope at the level of the cricoid. Varicose ulcers are rapidly fatal from hemorrhage; they are to be distinguished from peptic ulcer by the lack of previous pain and dysphagia; the presence of signs of cirrhosis of the liver points toward a varicose origin of the hemorrhage, but is not conclusive, as two cases of peptic ulcer have been complicated with cirrhosis. The other forms of ulcer usually lead to fibrous stenosis, and will be considered later.

Stenosis following peptic ulcer is to be distinguished from the following conditions: (1) Obstruction due to compression of the œsophagus from without; (2) stenosis due to causes other than peptic ulcer; (3) other diseases of the œsophagus accompanied by symptoms of obstruction.

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[1] See bibliography.

Among the causes of compression of the œsophagus from without, the most important are aneurysm, mediastinal tumors, and cold abscesses secondary to tuberculosis of the spine. Dysphagia, usually remittent, has been met with as a rare symptom in aneurysm of the transverse arch, and of the descending aorta; perforation into the œsophagus, an unusual accident, may be preceded by marked pain on swallowing, due to ulceration of the mucous membrane. The usual signs of aneurysm, and particularly the use of the *x*-rays, will make the recognition of such cases possible. Mediastinal tumors and cold abscesses, as sources of pressure, will usually become evident on a careful examination of the chest and spine.

True stenosis of the œsophagus may be divided into two classes, malignant and benign. Of these the former is by far the more common and important. On this account, and because several of the cases of peptic ulcer have been mistaken for cancer, it will be well to go somewhat in detail into the differential diagnosis between the two. The onset in cancer is more insidious, and the course of the obstruction more progressive, without such marked intermissions as are seen in ulcer, except in those rare cases in which the passage becomes open again after the sloughing away of part of the growth. The duration is short in cancer, usually not above six months after the appearance of marked symptoms, whereas the stenosis of ulcer may last for years. In cancer the hemorrhage is smaller in amount, consisting of a little blood mixed with food or mucus; exceptionally, however, one of the large vessels may be opened, in which case death soon takes place. The pain is somewhat similar to that of ulcer, but may occur at night, and independently of the taking of food. The situation is of importance, inasmuch as the stenosis due to ulcer almost never occurs so high as the bifurcation of the trachea, the favorite seat of cancer. A high obstruction, therefore, rules out ulcer, a low one may be due to either disease. Cachexia without obstruction for liquid as well as solid food speaks for cancer (Kraus).

Further points of importance in cancer are the presence of enlarged glands in the neck, and of pressure symptoms, especially paralysis of the recurrent laryngeal nerve. These do not occur in ulcer. The examination of the stomach contents is of little assistance, as anacidity may occur with either form of stricture. Pathognomonic for cancer are the findings with the œsophagoscope, and the microscopic appearance of fragments of the growth that may be withdrawn with the sound.

Stricture due to the action of corrosive poisons is readily distinguished by the history, and the presence, at times, of scars in the mouth or pharynx. The rare strictures following typhoid fever, scarlatina, and diphtheria come on during convalescence, and, therefore, are not likely to lead to error. Tuberculous and syphilitic strictures are extremely rare and are not preceded by such marked pain and hemorrhage as occur in peptic ulcer. The syphilitic variety is to be distinguished from peptic ulcer by its high situation near the pharynx, the presence of tertiary lesions elsewhere, and by the improvement under specific treatment, if instituted early. Stenosis following the ulceration due to foreign bodies is to be distinguished by the history; the presence of the foreign body may sometimes be detected by the sensation imparted on touching it with a stiff sound or by the use of the *x*-rays.

The œsophageal diseases likely to be confused with stenosis are nervous spasm, diffuse dilatation, and pulsion diverticulum. The first is usually situated at the cardia, but differs from the stenosis due to ulcer in the following points: solid food is often swallowed more readily than liquids, hemorrhage is lacking, and the obstruction, which can always be overcome by pressure, may disappear after a single passage of the sound. Signs of neurasthenia or hysteria are almost invariably present.

Diffuse dilatation which occurs as a result of cardiospasm, or of atony of the musculature of the œsophagus, may simulate stricture closely. It is recognized by the freedom with which the sound may be moved about in the œsophagus, the withdrawal from the latter of stagnating contents, in which no gastric ferments or hydrochloric acid are present, and by the œsophagoscopic findings. There is often an obstruction at the cardia, which yields when steady pressure is made.

Diverticulum is distinguished by its situation in the upper and back part of the œsophagus, the varying intensity of the obstruction, accompanied at times by a visible tumor in the neck, by the use of sounds (Leube's diverticulum sound), and by the œsophagoscopic appearances.

PROGNOSIS. Peptic ulcer of the œsophagus is a serious affection, not only from the danger of hemorrhage and perforation, but also owing to the marked tendency to the formation of stricture, which, though often yielding readily to dilatation, is very likely to recur. Furthermore, it is often a complication of grave diseases, which of themselves bear a bad prognosis. The number of cases diagnosed during life is as yet too small to justify any conclusions as to the exact mortality.

TREATMENT. The indications for treatment in the stage of ulceration are the same as for gastric ulcer. In order to provide conditions favorable for healing, irritation of the ulcer by the passage of food should be reduced to a minimum. Hence, rectal feeding will be in place for a short period, with rest in bed, to be followed by a Leube cure. All articles of diet which might cause mechanical, thermic, or chemical irritation are to be avoided. The general condition of the patient should receive careful attention.

Among drugs which, when given by the mouth, may have a direct action on the ulcer, silver nitrate ($\frac{1}{6}$ to $\frac{1}{2}$ grain, well diluted three times daily) and bismuth subnitrate (15 grains in suspension once daily) should be mentioned. It is probable that in some cases the drug will remain in contact with the ulcer long enough to exert a beneficial effect. Direct applications to the ulcer by means of the œsophagoscope probably will not be practicable on account of the danger of hemorrhage. Lavage of the œsophagus it also contraindicated for the same reason.

In the stage of stenosis, dilatation by means of bougies is usually necessary. This, in mild cases, may effect a complete cure; in others, the occasional passage of sounds must be kept up for a long time. When the stricture is impassable for sounds resort may be had to operative measures, usually gastrotomy, with subsequent feeding through the fistula. By this procedure not only is the nutrition rapidly improved, but the small ulcerations so frequently present at the stricture have a chance to heal, spasm ceases, and the œsophagus may become passable, as in Ewald's case.

In conclusion, I take pleasure in thanking Drs. R. H. Fitz, J. J. Minot, W. L. Richardson, and F. C. Shattuck, for their courtesy in placing the material at my disposal; and Drs. G. B. Magrath, F. B. Mallory, and J. H. Wright, for the use of the autopsy records.

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TABLE OF CASES REPORTED SINCE 1878.

Group I. Ulcer in Œsophagus Only.

No.	Author.	Sex; age.	Description of ulcer.	Microscopic examination.	Lesions in other organs.	Pain.	Dysphagia.	Hæmatemesis.	Duration.	Remarks.
1	Quincke	F. 41	Girdling ulcer from cardia to bifurcation.	Excludes cancer. (Langhans.)	Cancer of ovary; cancerous peritonitis; abortion.	General abdominal.	......	Present.	Eleven days.	
2	"	" 66	Numerous confluent ulcers in lower half; perforation into right pleura.	Excludes cancer.	Very large cyst of ovary.	......	......	"	A few days.	Perforation after tapping of cyst.
3	Sabel	" 32	Extensive, irregular, in lower third, exposing muscularis.	"Shows no cause for the ulcer."	Fibroma of ovary size of man's head.	Epigastric	......	"	......	Stools tarry.
4	Chiari	" 58	Extensive in lower half.	Excludes cancer and tuberculosis.	Incarcerated femoral hernia; perforation of bile duct; general peritonitis.	Present.	......	......	One week.	Blood in stomach at autopsy.
5	Shields	M. 66	Round ulcer, 1 cm. in diameter, in lower third.	......	Chronic interstitial nephritis; cirrhosis of liver; hypertrophy L. ventricle.	In front of chest.	Inconstant.	Coffee-grounds.	Six weeks.	
6	Reher	" 47	Girdling ulcer, 4.5 cm. broad, just above cardia; scar tissue reaching to 3 cm. above bifurcation.	......	......	In epigastrium and lower dorsal region.	Present; obstruction 36 cm. from teeth, due to diverticulum in ulcer.	Coffee-grounds.	Four years.	Free HCl present in vomitus.
7	Lindemann	" 48	Oval ulcer, 2x1 cm., just above cardia on posterior wall; edges thickened.	......	Double pneumothorax from perforation of ulcer; stomach normal.	Epigastric.	......	......	......	Epigastric pain, like that of ulcer, for several years.
8	Carstens	F. 25	"Chronic ulceration of lower half."	......	Tuberculosis of tubes, peritoneum, mesenteric glands; appendix abscess; pneumonia.	......	......	......	......	No history.
9	"	... ..	"Flat, peptic ulcers" in œsophagus.	......	Marked chronic interstitial nephritis: amyloidosis.	......	......	......	......	" "

Group I. (*Continued*).

No.	Author.	Sex; age.	Description of ulcer.	Microscopic examination.	Lesions in other organs.	Pain.	Dysphagia.	Hæmatemesis.	Duration.	Remarks.
10	Eversmann	M. middle aged.	Extensive; beginning 3 cm. above cardia; perforation into aorta; latter not sclerotic.	Excludes cancer.	Stomach full of blood.	In the chest, on swallowing and passage of sound.	Present.	......	Three and a half months.	Inconstant obstruction at cardia; sudden death from hemorrhage.
11	Fraenkel	M. 58	Stenosis just above cardia, with radiating scar tissue.	Excludes cancer.	......	...	Present.	......	One year.	Bartender.
12	Dasse	" 49	4 cm. above cardia in right postero-lateral wall, oval ulcer 2.5x 1.5 cm.	......	Aneurysm of aorta at fourth vertebra, adherent to œsophagus.	Slight burning in œsophagus on swallowing.	......	Profuse.	A few days.	Alcohol: "moderate."
13	Ortman	" 47	Carcinoma near cardia developing from scar of peptic ulcer.	......	Aspiration pneumonia, with gangrene of lung.	In lower end of gullet on swallowing.	Present.	Absent.	Two years.	Dysphagia first symptom ; improvement for one year with dilatation.
14	Rose	" 13	"Latent ulcer of œsophagus."	......	Perforation into pericardium; purulent pericarditis.	......	......	......	......	No history.
15	Janeway	F. one year.	Round, 1 cm. diameter, in L. lateral wall, 2.5 cm. above cardia; periœsophageal tissue inflamed.	......	"Infantile paralysis;" dysentery, pericarditis and pleurisy L.	......	......	......	......	No œsophageal symptoms.
16	Henoch	— five days.	Deep "ring-shaped" ulcer, 2 cm. diameter, with gray infiltrated base; situation, just above cardia.	......	Throat normal.	......	......	Present, also melæna.	Two days.	Melæna neonatorum.
17	Spiegelberg	M. five days.	Pea-sized hemorrhagic infiltration with erosion at junction of œsophagus and cardia.	Shows hemorrhagic infiltration, with necrosis.	......	......	......	Present, also melæna.	Two days.	Melæna neonatorum; blood cultures in life sterile.
18	Meyer	F. six days.	Small ulcer with thrombus adherent in R. wall just above cardia.	Inflammatory infiltration; streptococci present in ulcer.	......	......	......	Present, also melæna.	Three days.	Melæna neonatorum.

Group II. Ulcer at Cardia.

No.	Author.	Sex; age.	Description of ulcer.	Microscopic examination.	Lesions in other organs.	Pain.	Dysphagia.	Hæmatemesis.	Duration.	Remarks.
19	Quincke	M. 42	Ulcer at cardia extending 1.5 cm. into œsophagus; longitudinal scars above ulcer.	......	Ulcer also at pylorus; dilatation of stomach.	Present.	Present; inconstant obstruction at cardia.	......	......	Pocket formed at seat of ulcer, with valvular action.
20	Carstens	F. 2¾	" Large, perforating ulcer" at junction of œsophagus and cardia.	......	Tuberculosis of mesenteric and thoracic glands, intestines, peritoneum, tubes.	......	......	Stomach full of blood at autopsy.	......	No history
21	Eversmann	M. 47	Circular ulcer at cardia, extending into œsophagus and to posterior wall of stomach; scar tissue in places.	......	Perforation of that part of ulcer lying in stomach; adhesion to left lobe of liver, with necrosis and small abscess of latter.	On swallowing.	Present.	......	Five months.	Obstruction 42 cm. from teeth; perforation after sounding; clinical diagnosis, cancer.
22	Krokiewicz	" 46	Ulcer at cardia, extending into anterior wall of œsophagus.	......	Ulcers also at pylorus and lesser curvature; stomach dilated.	Between shoulder-blades.	Present.	Present.	Four weeks.	Free HCl increased; absent later.
23	Zaleski	" 39	Irregular ulcer of lower third of œsophagus and cardia, extending a short distance into stomach.	......	Scar of ulcer at pylorus with stenosis; dilatation of stomach; pulmonary tuberculosis.	Severe in epigastrium and lower sternum, radiating to back between shoulder-blades.	......	Present and cause of death.	Three months.	Free HCl diminished.
24	Fraenkel	" 47	Ulcer 4.5x6 cm. at junction of œsophagus and cardia; scar tissue in the base.	Chronic inflammatory tissue; gastric glands present in œsophagus.	....	Severe, epigastric, with tenderness.	......	Present.	Five months.	Free HCl present, 0.18 per cent.

Group III. Complicating Ulcers in Other Parts of the Stomach or in the Duodenum.

No.	Author.	Sex; age.	Description of ulcer.	Microscopic examination.	Lesions in other organs.	Pain.	Dysphagia.	Hæmatemesis.	Duration.	Remarks.
25	Quincke	M. 50	Stenosis due to contracted scar at lower end of œsophagus; hypertrophy above.	Inflammatory infiltration.	Scar of ulcer on posterior wall of stomach.	......	Present.	......	......	Gastric symptoms for "a considerable time."
26	Debove	" 55	Stenosis 5 cm. above cardia, due to scar; typical fibrous radiations.	......	Perforation of ulcer of anterior wall of stomach.	Epigastric, and at xiphoid, radiating to shoulder and eighth dorsal vertebra.	Present.	Present; repeated.	Five years?	Symptoms of gastric ulcer; eighteen years relief from dilatation; drunkard.
27	Zahn	" 46	Ulcer in lower œsophagus, with hypertrophy above.	Small-cell infiltration; thrombosis of an artery.	Ulcer of duodenum; dilatation of stomach; cirrhosis of liver.	Severe; epigastric.	......	Present; frequent.	Six weeks+	Drunkard; red cells, 950,000.
28	Vulpian	" 63	Six ulcers in lower fifth, involving almost entire circumference.	......	Smaller, shallower ulcers near pylorus.	Severe, epigastric of sudden onset.	Absent.	Absent.	Two months.	Severe dyspepsia five years before.
29	Ewald	" 30	Round; 3 cm. diameter, on R. wall just above cardia; perforation into lesser omental cavity.	Excludes cancer.	Duodenal ulcer with stenosis; pulmonary tuberculosis; localized peritonitis.	Severe epigastric, referable to duodenal ulcer.	......	Frequent.	Thirty days.	Free HCl present; stomach dilated.
30	Fraenkel	... ...	Round, with sharp, raised edges and funnel-shaped base, in lowest part of œsophagus.	......	Similar ulcer in duodenum.	......	......	......	......	Museum specimen of Rokitansky.
31	Kraus	M. 59	Girdling; extending 6 cm. upward from cardia.	Inflammatory infiltration, extravasation of blood.	Ulcer at pylorus; enormous dilatation of stomach.	......	......	......	......	Stools bloody.
32	Kraus	" 24	Numerous; confluent, extending to muscularis, situated in lower œsophagus; latter dilated.	Small-cell infiltration and extravasation of blood.	Scar of ulcer at duodenum, with stenosis; hour-glass stomach, with dilatation.	Epigastric, very severe.	......	Repeated; profuse.	......	
33	Hödlmoser	" 35	3.5 cm. in diameter; in lower end of œsophagus; anterior wall.		Two duodenal ulcers, with stenosis, and dilatation of stomach; hypoplasia of aorta.	At lower end of sternum; increased on taking food; tenderness.	Present.	Profuse; causing death.	Six weeks.	Symptoms of gastric ulcer five years before.

Group IV. Cases without Autopsy.

No.	Author	Sex; age.	Description of ulcer.	Microscopic examination.	Lesions in other organs.	Pain.	Dysphagia.	Hæmatemesis.	Duration.	Remarks.
34	Debove	M. 54	Two stenoses: (1) near cardia; (2) at level of top of sternum.	......	......	At sternum and between shoulder-blades.	Present.	Present; coffee-grounds.	Twelve years.	Alcoholic patient; cured by dilatation; no signs of syphilis.
35	Ortmann	" 27	Obstruction 40 cm. from teeth.	......	Stomach reaches 3 cm. below navel.	Epigastric.	Present.	Present.	Five years.	Sudden onset of dysphagia and pain; cured by dilatation.
36	"	" 30	Obstruction 43 cm. below teeth.	......	Stomach three fingers below navel; phthisis, third stage.	......	Present.	......	Four years.	Improved by dilatation.
37	"	F. adult.	Obstruction 32 cm. from teeth.	......	......	Paroxysmal at middle of sternum.	Present.	......	Several years.	Diagnosis by Quincke; improved by dilatation.
38	"	M. 42	Obstruction 42 cm. from teeth.	......	Ptosis of stomach.	At xiphoid.	Present.	Present.	Five years.	Free HCl present; relief from dilatation.
39	"	" 28	Obstruction 43 cm. from teeth.	......	Ptosis of stomach.	At xiphoid on swallowing.	Present.	Absent.	Five years.	Onset with violent pain in chest and stomach, after drinking sour wine; improved by dilatation.
40	Ewald	F. 19	Impassable stricture 30 cm. from teeth.	.....	Partial stenosis of pylorus.	At xiphoid on swallowing.	Present.	Absent.	Two months.	HCl increased; partial recovery after gastrostomy.
41	Starck	" 60	Ulcer, with dark red base and infiltrated edges, in left anterior quadrant, 38 cm. from teeth (œsophagoscope)	......	......	Between shoulder-blades.	Present.	......	Nine months.	Recovery.